## Letters to the Editor

### Collateral Aneurysms in Aortic Coarctation. A Contraindication for Percutaneous Intervention?

### *Aneurismas de la circulación colateral en la coartación aórtica, ¿son una contraindicación para el tratamiento percutáneo?*

**To the Editor,**

We have read with great interest the work published in *Revista Española de Cardiología* by Fernández González et al.[1] on the percutaneous treatment of patients with atretic aortic coarctation. We would like to make a number of comments on this article.

First, given the therapeutic challenges involved, we would like to congratulate the authors for their work in this subgroup of patients with atretic aortic coarctation. These patients typically have extensive collateral circulation and, due to the elevated pressure in the vessels, occasionally develop aneurysms that can become giant and compromise surgical access even further and increase perioperative risk.[2] Because the use of covered stents has decreased the incidence of aortic aneurysms after angioplasty and acute aortic injury,[3] many cardiologists prefer percutaneous treatment in patients with atretic aortic coarctation. Thus, the death of patient number 4 due to an aortoesophageal fistula is particularly interesting.

As described in the article, the patient was a 57-year-old man with a bicuspid aortic valve and aortic regurgitation who, as a preliminary step before valve surgery, underwent angioplasty of his aortic coarctation. The patient had developed extensive collateral circulation and a giant aneurysm of an intercostal artery. The procedure was completed without immediate complications and the patient had no residual gradient (from 70 to 0 mmHg). However, a fatal complication occurred at 48 hours. Strikingly, the autopsy revealed migration of the stent covering and consequent loss of the seal of the shunt from the intercostal artery (connecting the descending aorta and the aneurysm). The authors postulate that a postprocedural pressure increase in the descending aorta produced retrograde flow in the aneurysm that culminated in esophageal wall rupture.

Regarding the proposed pathophysiological mechanism, we would like to make some considerations: we question whether the increased retrograde pressure would induce aneurysm rupture, particularly when this aneurysm had already been subjected to a similar or greater anterograde pressure; in addition, after the disappearance of the gradient, the flow through the collateral artery, both anterograde and retrograde, would have decreased and partial or complete thrombosis of the aneurysm would be expected, which would contradict the theory that the rupture was due to excessive blood flow or high pressure; although stent coverings can help to prevent aneurysms of the aortic wall,[3] we do not consider sealing of the entire collateral circulation to be essential, given that the flow and pressure typically decrease, for the above reasons. Thus, we wonder where the stent covering ended up and why it became free. This last event was probably due to a product defect but we consider our first question to be vitally important because distal covering migration and flow obstruction could explain the acute and abrupt increase in proximal pressure or allow inflow into the aortic wall, which would produce local dissection and/or be directed toward the collateral circulation, affecting the aneurysm and promoting its rupture. We wonder whether the autopsy could shed further light on this matter.

Finally, we would like to state that, despite the unknown role played by these giant aneurysms in the treatment of these patients, their presence should not contraindicate percutaneous treatment. Indeed, we support this approach, given that, in our opinion, the complication in this patient was possibly more closely related to an obstruction caused by the loss and migration of the stent coating than to a technical fault and a loss of the seal of the distal orifice of the collateral artery.

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